Treating other effects of envenomation

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Introduction

Papuan taipans (*Oxyuranus scutellatus canni*), death adders (*Acanthophis* spp.) and Papuan blacksnakes (*Pseudechis papuanus*) are the most dangerous snakes along the Papuan coastline. Although the Papuan blacksnake is rare, the Papuan taipan is very common in parts of Milne Bay, Central, Gulf and Western provinces, and is the commonest venomous snake implicated in cases of snakebite. Deaths adders are widespread throughout mainland Papua New Guinea. The small-eyed snake (*Micropechis ikaheka*), brown snake (*Pseudonaja cf textilis*) and mulga snake (*Pseudechis cf. australis*) are also considered dangerous, but the incidence of bites by these snakes is not high and we know relatively little about them because not many studies have been done on these snakes in Papua New Guinea. Therefore, this section will mostly cover the effects of bites associated with the first three snakes. We will discuss the following effects:

1. Myocardial effects.
2. Rhabdomyolysis (muscle necrosis).
3. Acute renal failure.
4. Haematological effects.

We know that Papuan taipan (*Oxyuranus scutellatus canni*) venom contains other toxins apart from the major components, responsible for neurotoxicity and bleeding problems (coagulopathy). While death adder (*Acanthophis* spp.) venom is not known to cause any bleeding abnormalities, it may have other toxins, which may or may not be clinically significant; because there has been little research on PNG snake venoms, there is nothing much available in the current literature. The Papuan black (*Pseudechis papuanus*) has toxins causing neurotoxicity and a weak coagulopathy, which is associated more with platelet inhibition and anticoagulation than with thrombin activation and fibrinolysis.

Myocardial Effects

Symptoms and signs of myocardial effects

Rural health centres that lack the ability to monitor cardiac function by ECG monitoring or recording will not be able to detect some specific myocardial problems. It is important, however, to recognise some basic changes in cardiac function:

Bradycardia

The normal range for resting heart rate for an adult is 55-75 beats per minute (bpm). Generally speaking, a resting heart rate of less than 45-50bpm indicates bradycardia. The resting heart rate of a patient depends on the following factors:

- Age – faster in children and infants; faster again in older adults after middle age.

- Fitness – the resting heart rate is lower in fitter individuals.
- Co-morbidities (as below).

Moderate to severe bradycardia in an adult is indicated by a rate of less than 35 bpm (under 50bpm for a child, 80bpm for an infant). It is not necessarily a problem on its own, unless the peripheral perfusion is affected (end-organ perfusion), the blood pressure is low, or there is a clear conduction defect.

Tachycardia

A resting heart rate of more than 80 beats per minute indicates tachycardia in an otherwise well adult. Sinus tachycardia is common in snakebite patients, especially after Papuan taipan (*Oxyuranus scutellatus canni*) bite, and may be over 120bpm. Other contributing factors are:

- Anxiety.
- Co-morbidities – many acute and chronic medical conditions affect the resting heart rate; previous myocardial infarction, rheumatic heart disease and other valve disease; severe pulmonary disease, such as bronchiectasis, extensive pulmonary Tb, chronic obstructive lung disease; anaemia; dehydration from diarrhoea and vomiting; febrile illness and infections, eg. malaria, pneumonia; pregnancy.

Hypertension

The normal range for blood pressure for an adult is

- Systolic 100-130
- Diastolic 60-80

The exact pressure will depend on factors such as:

- Co-morbidities (as above).
- Age – lower in younger individuals.
- Sex – lower in women (SBP of 90 is considered normal in a young woman).
- Pregnancy – BP lower, especially in the second trimester.

A systolic blood pressure of more than 140-150/90 implies mild hypertension, 170-180/100 moderate hypertension, 200+/120 severe.

Hypotension

A diastolic pressure below 90 mmHg indicates hypotension, and in more severe cases the diastolic pressure can be less than 50 mmHg.

Electrocardiographic Findings

The commonest reported abnormal ECG findings are septal T-wave inversion, sinus bradycardia, atrioventricular block and other conduction defects. These are especially associated with bites by Papuan taipans (*Oxyuranus scutellatus canni*). Most venomous snakes in PNG may have myotoxic venom components capable of causing myocardial cell damage, but studies done have been inconclusive. The creatinine phosphokinase (CK) enzyme levels are high in some patients, but this is more likely to be due to rhabdomyolysis (skeletal muscle damage) than to myocardial damage. The cardiac enzyme troponin T, however, is a cardio-specific enzyme, which is released when heart muscle cells are damaged. It would seem likely that appreciable increases in the blood level of this enzyme would be indicative of

myocardial venom effects. However, serological studies from those patients with ECG abnormalities have shown no consistent relationship between troponin T and ECG changes.

There are some concepts put forward to explain the ECG changes seen in snakebite patients, but none of these have been universally accepted:

- Directly acting cardiotoxins (such as the myocardial Ca^{2+} channel blocker, taicatoxin) in venom causing myocardial damage and suppression of myocardial function.
- Myotoxins causing rhabdomyolysis and myocardial damage.
- Coronary vasospasm leading to myocardial ischaemia.
- Coronary artery thrombosis, especially in patients with a consumption coagulopathy, leading to coronary occlusion.
- Haemorrhagin (a toxin) causing myocardial haemorrhage.
- Electrolytes abnormalities causing electrical changes.
- Severe hypotension and/or respiratory failure causing hypoxia, leading to coronary insufficiency.
- Cardiac autonomic nervous supply disturbance.

A myotoxin (mulgatoxin) from the Australian mulga snake (*Pseudechis australis*) is known to cause both rhabdomyolysis and myocardial damage. A very closely related variant of this species occurs in Western province and neighbouring Papua. Papuan taipan (*Oxyuranus scutellatus canni*) venom does contain a potent voltage-dependent Ca^{2+} channel blocker (taicatoxin) that is specific to myocardial VDCC's as well as Ca^{2+}-dependent K^{+} channels in brain tissue. Whether or not this toxin is actually responsible for some of the electrocardiographic changes seen in taipan bite patients is unclear. Death adder (*Acanthophis* spp) venoms are unlikely to contain a similar toxin, but phospholipase A2 toxins are present in death adder venom, and the diversity of targets that these toxins have means that it is possible some might, one day, be found to cause myocardial effects.

Most experts believe that the ECG changes seen especially in patients bitten by taipans may be related to the toxin causing myocardial function suppression rather than direct damage. As explained above it may also be related to the actions of taicatoxin (calcium channel blocker) in taipan venom or to an abnormal autonomic nervous response to the general pathophysiology. The current belief is that taipan venom causes no direct myocardial damage.

Jensen (unpublished case) noted that a 35 year old man, who presented in respiratory failure more than 16 hours after a Papuan taipan (*Oxyuranus scutellatus canni*) bite, had an irregular, narrow complex (supraventricular) tachycardia of up to 140bpm, possibly atrial fibrillation, with a blood pressure in the normal range, and without apparent ST segment changes (no 12-lead ECG was available). This persisted after intubation, with satisfactory oxygenation and ventilation, opiate analgesia and sedation, and an initial ampoule of CSL polyvalent antivenom. A second dose, this time of CSL taipan antivenom, led to a fairly prompt reduction in the heart rate; within 1 hour he was in sinus rhythm at around 80bpm and remained so thereafter. No CK results are available because of a reagent shortage.

Treatment considerations

Rural health centres are not equipped to deal with serious electrocardiographic disturbances and patients with severe detectable rate and rhythm disturbances should be referred to a larger hospital, such as PMGH in Port Moresby, for treatment and monitoring.

Shock and hypovolaemic disturbances should be treated with appropriate IV fluids, preferably crystalloid (normal saline or Hartmanns), with urine output monitoring and central venous pressure monitoring if possible, if there is co-existent renal failure or other serious complication of envenoming.

Patients with marked bradycardia, especially with associated poor peripheral perfusion (can occur with a normal blood pressure) or hypotension can treated with atropine (0.02 – 0.05 mg/kg children; 0.3 - 3 mg adults). If persistent after IV fluid resuscitation, inotropic drugs such as dopamine may be useful for hypotension (also useful for bradycardic patients who do not respond to atropine), and vasodilators such as hydralazine or GTN might be useful for treating hypertension (though it is seldom severe, or persistent, enough to warrant intervention).

Rhabdomyolysis

Mulga snakes (*Pseudechis cf. australis*) and the small-eyed snake (*Micropechis ikaheka*) have specific myotoxins that cause rhabdomyolysis. In other species, phospholipase A_2 toxins have been long associated with rhabdomyolysis, leading to varying degrees of myoglobinuria, tubular necrosis of the kidneys and acute renal failure.

Rhabdomyolysis has been reported after bites by Papuan taipans (*Oxyuranus scutellatus canni*), and should be expected after bites by Papuan blacksnakes (*Pseudechis papuanus*). Although a species of death adder (*Acanthophis* spp.) from the highlands of Papua has been shown to produce a myotoxin (Acanmyotoxin 1), clinical myotoxicity has not yet been reported among patients bitten by death adders in Papua New Guinea.

Muscle cells contain a pigment called myoglobin that is similar to haemoglobin, although not the same functionally or structurally. This pigment is released from damaged muscle in large quantities and can cause indirect nephrotoxicity and renal tubular damage by blocking kidney nephrules, leading to acute renal failure via acute tubular necrosis, as well having direct tubular toxicity. Acute renal failure is a common complication in snakebite patients admitted to PMGH.

Elevated creatinine phosphokinase (another enzyme liberated from inside damaged muscle cells) is elevated in many snakebite patients and is a clear indication that some muscle damage is present. There are also variants of this enzyme liberated when the myocardium is damaged, and without knowing exactly which isoenzyme variant is being released, this can lead to confusion over whether or not the damage is skeletomuscular or myocardial in nature. If troponin T levels are normal in the presence of elevated CK, then rhabdomyolysis is the likely cause.

What is the clinical importance of rhabdomyolysis in snakebite patients?

1. Rhabdomyolysis causes myoglobinaemia and myoglobinuria, resulting in kidney damage that leads to a degree of acute renal failure – a potentially life-threatening condition.
2. Electrolyte abnormalities also occur and these can have very serious, mainly cardiac effects.
3. Massive muscle necrosis can occur and may be debilitating, prolonging recovery, which is however, eventual.

Symptoms of Rhabdomyolysis

- Generalized muscle pain or tenderness ('aching muscles') is usually reported by the patient within 3-4 hours of the snakebite. In the case of the Papuan blacksnake (*Pseudechis papuanus)* there may also be bite site pain.
- Weakness of the limbs (care that this might indicate a degree of neurotoxicity, so check for ptosis as well).
- Complaints of discoloured urine.
- There may also be bilateral renal angle pain.

Signs of Rhabdomyolysis

From a practical perspective, the following may be the only detectable signs:

- Discoloured dark brown urine (often described as being 'Coca cola coloured') that may appear 'stringy' if left to stand; will test positive for blood on the urine dipstick, with no red cells seen in the urine on microscopy (unless there is co-existent coagulopathy).
- Proteinuria (measured by 'dipstick').
- Hyperkalaemia (elevated serum potassium level), with ECG changes if the level rises over 7 mmol/l – tall, peaked T-waves and a prolongation of the QRS duration beyond 0.12 s; progresses to SA node and AV node impairment with bradycardias or life-threatening ventricular dysrhythmias as the level rises much further.
- Fluid imbalance: a significant reduction in urine output when there is adequate fluid input (by IV or by mouth), blood pressure and oxygenation, may be indicative of renal impairment.

Laboratory findings

Many of the important tests for rhabdomyolysis are unavailable in PNG at the present time, and certainly in rural health centres, definitive tests are not available. If tests are available, the following indicate rhabdomyolysis:

- Elevated creatine phosphokinase (CK) levels (levels > 250 IU/L)
- Elevated serum creatinine levels (an increase > 0.2 mmol/L)
- Elevated serum myoglobin (levels > 80 ng/ml)
- Elevated aspartate transaminase (AST) (levels >50 IU/L)

Treatment

The most important treatment of snake venom-induced rhabdomyolysis is the neutralisation of the circulating toxins with appropriate antivenom.

Very little other treatment is possible in a rural health centres, although it is important to:

- Maintain proper fluid load and hydration, but avoid fluid overload, which can lead to pulmonary oedema, even without co-existent renal failure (this may be indicated if a patient receiving high volumes of fluids becomes breathless); up to 1000 ml/hr for the first 4 hours, then reduce to a level that gives the required urine output.
- Maintain a urine pH>7 (this may be on your urine dipsticks), since myoglobin is more toxic at a lower pH; 50 mmol/hr for the first hour, then 30 mmol/hr; beware that this can worsen hypocalcaemia, resulting in abdominal pain, skeletal muscle fasciculations,

convulsions, and ECG changes (prolonged QT interval) due to severe hypocalcaemia in patients whose underlying cause of renal impairment is rhabdomyolysis.

- If fluid replacement (and blood pressure and oxygenation) is adequate, but moderate to severe renal impairment is present (oliguric urine output < 0.5 ml/kg/hour) give 40 mg frusemide over 2-3 minutes. If urine output does not improve to at least 40 ml/hour, then give a second dose of 100 mg over 20 minutes; a third dose of 200 mg over 40 minutes may be given is there is certainty about the adequacy of the fluid status of the patient, but persistent oliguria, or the patient is likely to have pulmonary oedema (and not pulmonary aspiration). Ideally the urine output should be maintained at 2 ml/kg/hour.
- If frusemide does not improve urine output, then a **single dose** of 200 ml of 20% mannitol IV over 20 minutes may be given. It is important that you **do not give more than one dose** of mannitol, as this can precipitate dangerous fluid electrolyte imbalances once it causes a diuresis.

Additional considerations

In Papua New Guinea, an observation of dark coloured urine is often interpreted as haematuria or haemoglobinuria, when myoglobinuria is more likely. There are no dipstick tests that distinguish directly between haemoglobinuria and myoglobinuria; however, if muscle weakness, pain or tenderness is reported, then myoglobinuria is the more likely diagnosis.

In facilities with a microscope, haematuria (red cells in the urine) can be confirmed by examination of a centrifuged sample under the microscope.

It is important to understand that patients with profound rhabdomyolysis may take many weeks to recover their muscle strength, and that while physiotherapy during the course of the myolysis can be extremely painful, physiotherapy during recovery is important to healing.

Acute Renal Failure

Acute renal failure with electrolyte imbalance is a major cause of morbidity and prolonged hospital stay in snakebite patients. In fact, a high percentage of snakebite patients develop renal failure, with some requiring peritoneal dialysis. Snake venoms can induce renal failure in several ways:

- Direct nephrotoxicity: venom can directly damage the renal glomeruli and tubules, and patients develop acute tubular necrosis and renal failure.
- Myoglobinuria, as discussed above, can also cause acute renal failure.
- Immune-mediated renal damage, especially by the humoral mechanism, is also possible.
- Pre-hospital renal failure is likely in those patients who are kept in the rural health centres on a nil-by-mouth regime without intravenous fluids, and also in those who develop renal insufficiency secondary to prolonged hypotension or hypoxia. Snakebite patients need good hydration in order to maintain an optimal urine output and renal function. Snake venom toxins are also cleared from the body in urine, so a higher output may help to remove circulating toxins.
- In those patients with a severe coagulopathy (defibrination syndrome or DIC), fibrin strands and fibrin degradation product (FDP) deposition in glomeruli, or thrombosis of renal vessels, can cause renal failure.

Acute renal failure is a serious, potentially life-threatening condition. In the study of snakebite mortality that was conducted among patients admitted to the PMGH ICU between January

1992 and December 2001, renal failure was a direct cause of death in 10% of the cases, and was a contributing factor in another 16.7% of fatalities.

Patients with moderate to severe renal failure need urgent medical care in a high dependency or intensive care facility, and patients who develop signs and symptoms of renal failure in a rural health centre should be transported to a larger hospital as soon as possible.

Symptoms and signs of developing renal failure

In a rural health centre setting, where there are no laboratory facilities, the diagnosis of renal impairment is seldom straightforward. The following symptoms may indicate a developing renal insufficiency:

- A greatly reduced urine output despite adequate hydration, oxygenation and blood pressure and the use of an IDC. A satisfactory output is >35-45 ml/hour; if the output drops to < 0.5 ml/kg/hr, this indicates oliguric renal impairment. Anuria is a complete lack of urine production, and therefore a very serious condition.
- Development of a syndrome of clinical uraemia featuring:
  - ? hiccups;
  - ? nausea and vomiting;
  - ? drowsiness;
  - ? confusion;
  - ? coma;
  - ? muscle twitching, 'flapping' tremor or convulsions;
  - ? pericardial friction rub or evidence of a pericardial effusion;
  - ? development of fluid overload;
  - ? symptoms suggestive of hyperkalaemia, such as arrhythmias.
- Hypovolaemia: defined by postural hypotension (a reduction in BP upon standing; supine hypotension comes later), cool peripheries, sunken eyeballs (especially children), dryness of the mucosal tissues, loss of skin turgor and 'empty' neck veins.

Treatment in a rural health centre

A patient who is developing renal failure needs to be evacuated to a larger medical facility as a matter of priority.

There are some steps that can be taken at the health centre that may be helpful:

- Fluid challenge: Give 1-2 litres of isotonic (normal) saline over 1-2 hours with close observation of their BP, jugular venous pressure and respiratory rate. Stop the challenge if the vertical height of jugular venous pulsation reaches 6-8 cm above the sternal angle, with the patient reclining at 45 degrees, or if respiratory distress develops.
- After the fluid challenge, continue to maintain an adequate fluid load and hydration, but **avoid fluid overload,** which can lead to pulmonary oedema and other serious complications (this may be indicated by breathlessness in a patient receiving a large volume of fluids).
- Correct hyperkalaemia: bradycardia (or rapid tachycardia) and/or shortness of breath – implies respiratory compensation for a metabolic acidosis, which is usually accompanied by hyperkalemia; there may also be accompanying pulmonary oedema; the following medications should be given/commenced in this order:
  - ? 10 ml of 10% calcium gluconate IV over 2 minutes, while monitoring heart rate and rhythm; this dose can be repeated up to 3 times every 30-60 minutes.

- ? 50 ml of 50% dextrose, with 10 units of soluble insulin (may not be required in the patient who does not have diabetes), IV over 1 hour; monitor the blood sugar hourly for the next 4 hours.
- ? 1 ml/kg (1 mmol/kg) of 8.4% sodium bicarbonate given **slowly** over 30 minutes, but be cautious as this may cause abdominal pain, skeletal muscle fasciculations, convulsions, and ECG changes (prolonged QT interval) due to severe hypocalcaemia in patients whose underlying cause of renal impairment is rhabdomyolysis.
- ? Salbutamol, 5-20 mg nebulised (or by Ventolin inhaler).
- ? Increase the IV fluid load (provided the patient is making SOME urine), accompanied by IV frusemide (40-80 mg IV over 5-20 minutes).
- ? Rectal or oral sodium resonium resin will be required (10-15 g tds).
- ? Stop any K+-containing medications or infusions, and restrict dietary intake of K+ (eg. bananas); stop K+-sparing diuretics.

- If fluid replacement (and blood pressure and oxygenation) is adequate, but moderate to severe renal impairment is present (oliguric urine output < 0.5 ml/kg/hour) give 40mg frusemide over 2-3 minutes. If urine output does not improve to at least 40 ml/hour, then give a second dose of 100mg over 20 minutes; a third dose of 200 mg over 40 minutes may be given is there is certainty about the adequacy of the fluid status of the patient, but persistent oliguria, or the patient is likely to have pulmonary oedema (and not pulmonary aspiration). Ideally the urine output should be maintained at 1-2 ml/kg/hour.
- If frusemide does not improve urine output, then a **single dose** of 200 ml of 20% mannitol IV over 20 minutes may be given. It is important that you do not give more than one dose of mannitol as this can precipitate dangerous fluid electrolyte imbalances once a diuresis occurs.

Haematological Effects

Coagulopathy and neurotoxicity are the most common pathophysiological changes seen in most snakebite patients in Papua New Guinea, and they are covered widely in this Handbook by other authors. Apart from coagulopathy, snake venoms may also cause other pathophysiological changes in the blood and blood vessels:

1. Haemolysis (breakdown of red blood cells).
   (a) Microangiopathic haemolysis: Destruction of red blood cells in the microvasculature (capillaries and arterioles) due to a change in their shape and size brought about by venom components. Prothrombin activation and fibrinolysis, common in Papuan taipan envenomation, leads to accumulation of fibrin degradation products (FDP). The deposition of fibrin strands in small vessels can contribute to haemolysis by catching red cells as they pass.
   (b) Direct attack on the red blood cell phospholipid bilayer, leading to cell damage and haemolysis. Sphero-echinocytic changes in red cell morphology have been observed after a taipan bite in Australia and may be due to either phospholipase A_2 or phospholipase B.

Haemolysis is evident by a falling haemoglobin level, increased bilirubin level and haemoglobinuria.

2. Platelet abnormalities:
   (a) Vascular endothelial damage, leading to microthrombosis and platelet consumption, may occur after the bites by some Papua New Guinean snakes.

(b) Most patients have a normal platelet count, but if they continue to bleed, this may suggest that some component of the venom toxin is suppressing the normal function of the platelets. Platelet dysfunction is a common concomitant finding in snakebite patients with coagulopathy.
(c) Thrombocytopenia, due to platelet aggregation inhibition and persistent bleeding, may be occur in some snakebite patients many hours after a bite, and may be first noticed when the patient experiences epistaxis.

Treatment issues

The issue of using blood products in the treatment of coagulopathy is dealt with in Chapter 9.

Heparin and antifibrinolytic drugs have no place in the treatment of coagulopathy in Papua New Guinea. Heparin may precipitate further bleeding and must not be used.

Local effects of snakebites

The majority of patients with snakebite experience few if any local effects from the bite itself.

Local pain

Local pain has been reported after bites by death adders (*Acanthophis* spp.) and is a particular feature of bites by blacksnakes (*Pseudechis* spp.) in Australia, although it is poorly documented in Papuan blacksnake (*Pseudechis papuanus*) envenomation. Local pain and swelling has been reported after envenomation by the small-eyed snake (*Micropechis ikaheka*).

If necessary, pain management should be dealt with cautiously. Sedatives or other agents that affect consciousness should be avoided if possible. If pain medication is considered necessary then consider:

- Paracetamol suppositories or crushed tablets via an orogastric tube.
- Codeine.

Non-steroidal anti-inflammatory analgesics, especially aspirin, should be avoided in the case of snakebite where coagulopathy may occur, or is already present.

Local tissue effects

Some degree of swelling, oedema, and ecchymoses may be present after the bites of Papua New Guinean snakes. Bruising to the bite site may be either the result of the trauma caused by the bite itself, especially if the snake was large. Small-eyed snakes (*Micropechis ikaheka*) may bite and grip the victim for some time before either letting go, or being kicked free. In these cases the bite site bruising may be distinctive.

Early coagulopathy may also result in local ecchymoses at the bite site, but is often associated with oozing of blood from the punctures. Mild to moderate localised swelling may cause discomfort that can be best managed with paracetamol, elevation and cold packs.

Most snake venom related swelling or oedema should subside within 5-7 days without requiring further treatment. In a few cases more significant local injury may occur, including the formation of haemorrhagic bullae containing lysed cells and blood. This has been seen after bites by Australian species of blacksnake (Pseudechis spp.) and may therefore also occur

after bites by either Papuan blacksnakes (*Pseudechis papuanus*) or New Guinean mulga snakes (*Pseudechis cf. australis*).

Local swelling and oedema may also result from ischaemic injury caused by the prolonged use of homemade tourniquets from materials such as rubber tyre inner-tubes, grass fronds, rope or electrical wire. Very severe cases of ischaemic injury can lead to amputation.

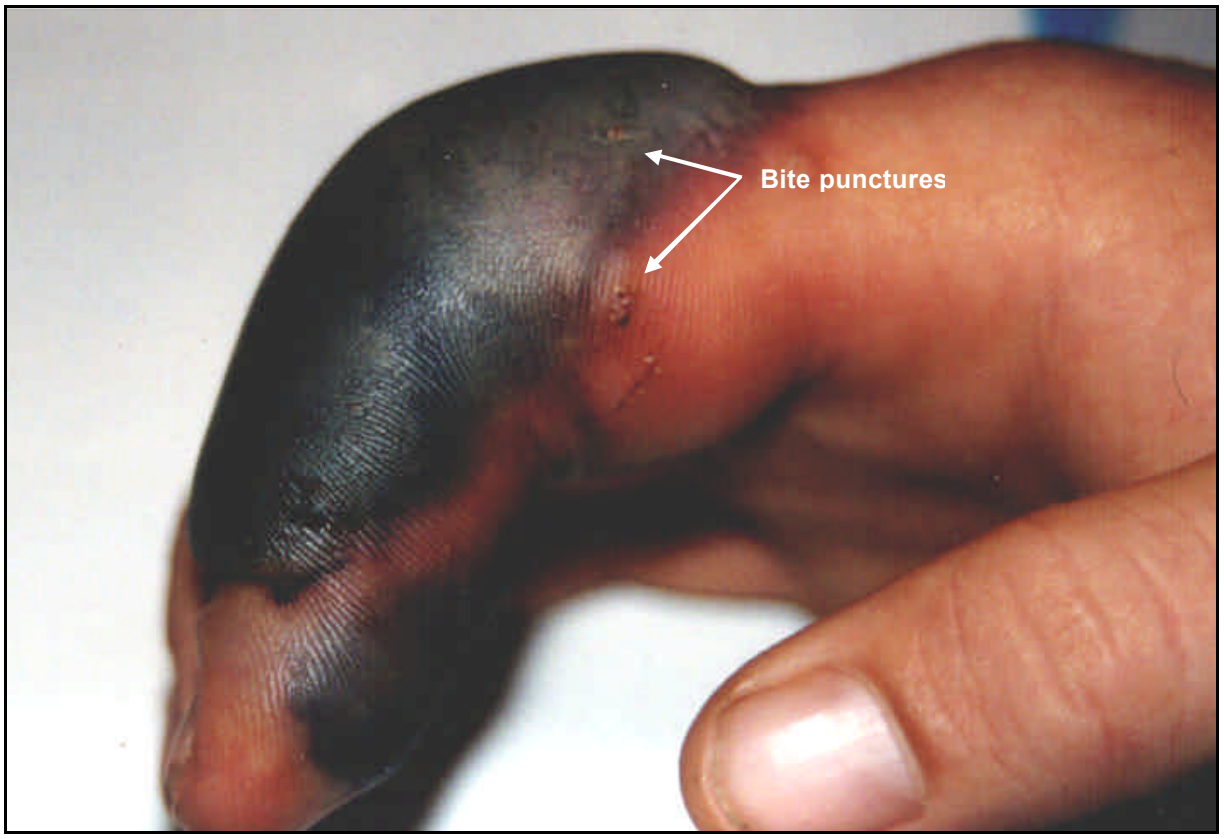

FIGURE 1: Local swelling, ecchymoses and haemorrhagic bullae after the bite of a 30 centimetre spotted blacksnake (*Pseudechis guttatus*) from Australia. It is possible that similar local injury could result from bites by Papuan blacksnakes (*Pseudechis papuanus*) or New Guinean mulga snakes (*Pseudechis cf. australis*). Treatment of this case involved surgical debridement of tissue

Secondary infection and sepsis

Clinical infection at a bite site is rarely present by the time a patient presents for medical attention. However, there is a tradition in PNG of giving every snakebite patient, and, in fact, every patient with trauma (sometimes even with blunt trauma), penicillin, and sometimes chloramphenicol as well. This may be in the form of a stat dose, or ongoing treatment for several days until someone notices and decides to cancel the order.

Various theories have been presented as to why this tradition exists. While there might be some sympathy for hope that this helps to prevent a subclinical infection developing into a clinical one (this is an animal bite, after all), there seems to be no research to support this. It is perhaps more likely is that there will be selective growth of resistant organisms. Certainly the excessive use of any antibiotic in the past, around the world, has lead to the development of

antibiotic resistance. Similarly, there seems to be little logic, and no research, to support the idea that a single dose of penicillin will help to prevent the development of tetanus infection, though a tetanus vaccination soon after the bite might do so. Tetanus infection develops after germination of infecting spores, which are not sensitive to penicillin. Another adverse result of giving antibiotics unnecessarily is that the time, syringe, needle and dose of antibiotic are not being used for another patient who might stand to benefit from them more.

There are some special circumstances in which antibiotic therapy should be considered in snakebite:

- Established cellulitis around the bite site.
- A retained foreign body, eg. a fang, imbedded in the skin, indicating a deeper inoculation.
- Early collapse with a compound fracture, or a large laceration.
- Aspiration pneumonia (pulmonary aspiration with a fever).

Ideally, suitable microbiological samples should be obtained for culture before antibiotic therapy is commenced. If antibiotics are given, they should be given intravenously (or orally or via the NGT), not intramuscularly, especially when the patient is known to have a coagulopathy.

As with any wound, local wound care is **very** important in the prevention of infection, which is due not only to introduced bacteria, but also to local skin flora. This care should probably be delayed in the snakebite patient until they are at a facility where they can be given, and have been given, antivenom, where this is indicated. The basis for this assertion is that local cleaning may release more venom into the circulation; pressure bandaging should certainly not be removed to clean a wound.

Once it has been established that a patient has not been envenomed, or has been given the correct antivenom, and any coagulopathy has begun to resolve, the wound may be cleaned with saline, all foreign material removed, and Betadine applied to the skin of the immediate area. The wound will generally not need dressing, provided the patient maintains cleanliness of the area for the next few days. This procedure is very effective in preventing wound infections in other types of wound, and there is no reason to believe that it would not be applicable to snakebite as well. However, its efficacy compared to no wound care at all, which is the norm in PNG, has not yet been formally studied.

If a patient has not had a full tetanus vaccination course, and suffers snakebite, or any other trauma resulting in a break in the skin, they should receive a booster. If the person has never received the vaccination, they should be encouraged to have a full 3-dose course, with the subsequent doses being at 6 weeks and 6 months after the first.

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